Antiretroviral therapy (ART) for HIV is not a cure. However, recent studies suggest that ART, initiated early during primary infection, may induce post-treatment control (PTC) of HIV infection with HIV RNA maintained at <50 copies/ml. We investigate the hypothesis that ART, initiated early, permits PTC by limiting the size of the latent reservoir, which if small enough at treatment termination, may allow immune responses to prevent viral rebound (VR) and control infection.

We use an ODE model of within host HIV dynamics to capture interactions between target cells, productively/latently infected cells, virus, and cytotoxic T lymphocytes (CTLs). Bifurcation analysis reveals a range in CTL response strengths where viral loads exhibit bistability between a high viral set point (VR) and a low viral set point (PTC). Below the bistable range, patients will always rebound, while above the range patients are predicted to behave like elite controllers. We show the basins of attraction associated with the VR and PTC viral set points and their dependence on the latent reservoir size at treatment termination.

Using data on latent reservoir sizes in patients treated during primary infection, we also predict population-level VR times for non-controllers consistent with observations. (Received January 17, 2015)